against smooth muscle alpha actin. Capillary endothelial cells (stained with an antibody against von Willebrand factor, vWF) showed only weak colocalization with bFGF so that the relationship between the morphometric area fractions of vWF and bFGF was even inverse ($R = -0.662$, $p = 0.0005$). Similarly to bFGF, intense immunolabeling of platelet derived growth factors AA and BB (PDGFs) was observed on vascular smooth muscle cells, whereas its correlation with the vWF-staining of capillary endothelial cells was inverse ($R = -0.387$, $p < 0.025$). PDGFs were not detected in the extracellular matrix. In contrast to bFGF and PDGFs, transforming growth factor-β (TGF-β) was primarily associated with vWF-positive capillary endothelial cells. The extracellular matrix contained TGF-β in areas of scarring and repair. *Conclusion:* In the failing myocardium, TGFβ is primarily expressed on the capillary endothelium, wheras PDGFs and bFGF are more associated with the vascular smooth muscle cells of intramural coronary arteries. In addition, large amounts of bFGF are bound to collagen type 1. These distinct patterns of distribution of growth factors suggest a network of feedback mechanisms involving different cell types and the extracellular matrix during ventricular remodeling.

719 Syncope and Sudden Cardiac Death: Clinical Predictors, Tilt Testing, and Electrophysiologic Testing

Monday, March 20, 1995, 2:00 p.m.–3:30 p.m.
Ernest N. Morial Convention Center, Room 90

2:00

719-1 Does Vasodepressor Syncope Require Normal Ventricular Function?

Andrea Natale, Jasbir Sra, Anwer Dhala, Michael Biehl, Abdul Wase, Sanjay Deshpande, Cheryl Maglio, Margaret Budziszewski, Masood Akhtar. *Sinai Samaritan/St. Luke's Medical Center, Milwaukee, WI*

Vasodepressor syncope is believed to be preceded by hypercontractility which leads to mechanoreceptor stimulation. Therefore, a preserved left ventricular function is supposedly required to trigger the reflex. We analyzed our experience in patients with positive head-up tilt test. Out of 500 patients 32 had previous history of structural heart disease. Of them 8 patients had severe left ventricular dysfunction (EF < 30%). The mean age was 52 ± 8 years, 5 were male and 3 female. Mean EF was 22 ± 5%. Six patients had dilated cardiomyopathy and 2 had myocardial infarction. Electrophysiologic study was negative in 6 patients while the remaining two had inducible sustained monomorphic ventricular tachycardia requiring defibrillator implant. Syncope was witnessed by physicians in 6 out of 8 patients and was associated with bradycardia and hypotension. In all 8 patients head-up tilt reproduced symptoms. Upright tilt was positive at baseline in 7 patients and during isoproterenol infusion in 1 patient. Five patients were treated with theophylline, 1 with ephedrine, and 2 with disopyramide. After a mean follow-up of 2.2 ± 1.3 years all 8 patients were free of vasodepressor-related syncope.

Conclusion: We conclude that vasodepressor syncope is possible even in the presence of severely impaired ventricular function and should, therefore, be entertained in such cases. Alternative mechanisms may mediate the circulatory response in this population.

2:15

719-2 Evolution of Patients with Neurocardiogenic (Vasovagal) Syncope not Subjected to a Specific Treatment

Aurora Ruiz, Adelqui Peralta, Eleonora Duce, Jorge Scaglione, Jorge González-Zuelgaray. *Argerich Hospital, Buenos Aires, Argentina*

The results obtained with some drugs or other therapeutic strategies in the prevention of neurocardiogenic syncope are encouraging. However, the benefit they represent when compared with the natural history of this disorder has not been clearly established. The purpose of this prospective study was to analyze the natural history of patients with neurocardiogenic syncope.

Fifty-six consecutive patients with either: a) One or more episodes of syncope and a positive head-up Tilt test (29 patients), or b) Typical history of neurocardiogenic (vasovagal) syncope (27 patients) despite a negative Tilt test, were followed up by telephone or personal consultation during a mean period of 16.1 ± 10.2 months. Patients were discharged without any specific medication.

There were no deaths during the follow-up period. Recurrences were found in 5 patients (8.9%). The risk of recurrence was 7% after one year and 15% after 21 months. The multivariate analysis showed the duration of symptoms before the inclusion as an independent variable related to recurrences ($p < 0.025$).

Conclusions: The prognosis of patients with neurocardiogenic syncope is excellent as far as survival is concerned. Recurrences of episodes without any specific treatment are uncommon after diagnosis has taken place. This finding strengthens the need to evaluate the effect of drugs with placebo control groups and to select the highest risk group for treatment.

2:30

719-3 Usefulness of Signal-Averaged ECG, Head-up Tilt and Electrophysiologic Studies in Patients with Unexplained Syncope

Carlos A. Morillo, Sally Zandri, George J. Klein, Raymond Yee. *McGuire VA Medical Center, Richmond, VA; University Hospital, London, Ontario, Canada*

The recent availability of non-invasive diagnostic methods such as signal-averaged ECG (SAECG) and Head-up tilt (HUT) have raised the need to evaluate the role of such tests for the screening of pts with recurrent unexplained syncope (US). The purpose of the present study was to identify the clinical variables that predicted an abnormal SAECG and HUT in a series of 70 consecutive pts. referred for assessment of recurrent US. SAECG (ART EPX 1200) and HUT @ 60° with a low-dose isoproterenol protocol for 30 minutes, were performed in all pts. SAECG was considered (+) if ≥2 of Simson criteria were abnormal. A (+) HUT was defined as the induction of presyncope or syncope associated with bradycardia and hypotension that resembled the clinical episode. All pts underwent 2D-echo, radionuclide angiography and electrophysiologic study (EPS).

VT(+)	SAECG(+)	SAECG(−)	HUT(+)	HUT(−)	EPS
N Pts	24 (34%)	46 (64%)	30 (42%)	40 (57%)	29 (41%)
AGE	42 ± 12	45 ± 16	36 ± 10*	55 ± 10*	45 ± 15
EF%	34 ± 8*	53 ± 6	55 ± 9	53 ± 5	36 ± 5*
PMI	16 (67%)*	4 (9%)	2 (6.6%)	2 (5%)	19 (66%)*

EF = Eyection fraction, PMI = Previous myocardial infarction, * = Multivariable analysis $p < 0.01$

A (+) SAECG, previous MI and EF < 40% were highly predictive (95%) of induction of sustained monomorphic VT during EPS. Similarly, (−) SAECG and age < 50 yrs were associated with a (+) HUT in 96%. Overall a cause for US was determined in 77%.

Conclusions: A (+) SAECG, EF < 40% and history of a previous MI were highly predictive of the induction of VT during EPS in pts with US. A (−) SAECG, EF > 40% and age > 50 yrs were predictive of a (+) HUT. Non-invasive screening of pts with US with SAECG and HUT increased the diagnostic yield and should be performed prior to invasive assessment with EPS.

2:45

719-4 RR Variability Preceding Head Up Tilt Predicts Syncope in Children

Julian M. Stewart, Markus Erb, David Rubin, Carmine Sorbera. *New York Medical College, Valhalla, NY*

To assess RR variability during head up tilt (HUT) we studied 20 syncopal pts aged 7–18y with Holter monitor. After 30 min supine, pts were tilted to 80° for 30 min or until syncope occurred. Sequential 256 and 512 beat epochs free from ectopy were analyzed. Mean and standard deviation, root mean square (RMSSD), fraction exceeding 50 msec (pN50) and correlation coefficient (r) of successive differences were computed. An autoregressive power spectrum model was used. Low frequency power (LFP, 0.05–0.15 Hz), high frequency power (HFP, 0.15–0.40 Hz), and total power (TP, 0.01–0.40 Hz) were compared. Epochs while supine, within 5 minutes after HUT, 5–10 min after HUT, and 15 min after or during syncope were analyzed. 12 pts fainted (F) and 8 did not (N). RR interval was 910 ± 6 for N and 870 ± 4 msec for F (p = NS). Indices of RR variability were markedly different ($p < 0.05$) for F and N while supine: RMSSD was 128 ± 22 for N vs 62 ± 7 msec for F, pN50 was 0.51 ± 0.06 for N vs 0.33 ± 0.04 for F, r was 0.50 ± 0.06 for N vs 0.70 ± 0.05 for F. LFP was 920 ± 140 in N vs 1710 ± 440 msec^2 in F (p = NS), while HFP and TP were 2990 ± 840 and 7062 ± 1500 in N vs 834 ± 130 and 2855 ± 420 msec^2 in F ($p < 0.05$). With HUT RR decreased similarly in F and N (25 ± 3% and 26 ± 3%) while RMSSD and pN50 decreased proportionately in N (by 59 ± 5% and 63 ± 10%) and in F (by 59 ± 4% and 83 ± 5%, p = NS). RMSSD and pN50 remained higher in N vs F after tilt. r increased for N (to 0.77 ± 0.05) and F (to 0.88 ± 0.02) but remained higher in F reflecting lower variability. LFP, HFP, and TP decreased proportionately for each group. Thus HFP and TP remained higher in N than F. Similar results were obtained after 5–10 minutes of HUT in N and F and after 15 minutes in N. In F prior to syncope, RR increased to 1,045 ± 50 msec, RMSSD to 125 ± 25 msec, PN50 to 0.41 ± 0.05, and r fell to 0.51 ± 0.04, while TP increased to 5200 ± 1343 msec^2, LFP increased to 1717 ± 601 msec^2, and HFP increased to 2730 ± 1075 msec^2. Relatively decreased RR variability supine

prior to HUT predicts syncope and is primarily caused by decreased high frequency power.

3:00

719-5 Clinical Correlates and Prognostic Significance of the Frequency of Episodes of Nonsustained Ventricular Tachycardia in the Electrophysiologic Study versus Electrocardiographic Monitoring Trial (ESVEM)

Kelley P. Anderson, Motomi Mori, ESVEM Investigators. *University of Pittsburgh, Pittsburgh, PA*

Nonsustained ventricular tachycardia (VTns) predicts mortality in several settings but its significance in patients (pts) with a history of sustained ventricular tachyarrhythmias is unknown. Pts in ESVEM were randomized to guidance method and to drug independent of VTns events. We grouped pts (n = 486) by frequency of VTns events on baseline 48hr drug-free HM: G1 ≡ No VTns (n = 70), G2 ≡ >0 to <0.25 runs/h (n = 190), G3 ≡ ≥0.25 to <1 runs/h (n = 109), G4 ≡ ≥1 runs/h (n = 117). There were no significant differences ($p > 0.05$) between groups with respect to age, ejection fraction, functional class, presenting arrhythmia, prior exposure to antiarrhythmic drugs or guidance method. Data are presented in order of group number (Gr1, 2, 3, 4, respectively). Significant differences were observed for sex (29, 8, 13, 15 %female; $p = 0.0008$), previous myocardial infarction (6%, 10%, 22%, 23%; $p = 0.0002$), PVC/h (136, 146, 300, 735; $p < 0.0001$), pairs/h (1, 3, 12, 60; $p < 0.0001$), and mean heart rate (77, 74, 74, 80 bpm; $p = 0.0008$). Because the significance of VTns may vary in pts with different disorders, an analysis was restricted to pts with ischemic heart disease (IHD, n = 414). Significant differences were observed for age (63, 65, 64, 67 yr; $p = 0.01$), sex (23, 6, 10, 9 %female, $p = 0.0028$), years since last myocardial infarction (MI) (3.7, 8.9, 9.1, 7.9yr; $p < 0.0001$), history of operative revascularization (8%, 29%, 30%, 38%; $p = 0.0003$), for PVC/h (151, 149, 286, 769 PVC/h; $p < 0.0001$), mean pairs/h (1, 3, 12, 62; $p < 0.0001$), mean heart rate (76, 74, 76, 81bpm; $p = 0.004$), but not for ejection fraction, functional class, presenting arrhythmia, previous exposure to antiarrhythmic drugs, number of MIs, or angina since last MI. Variables based on presence/absence, on continuous and on stratified frequency measures of VTns events and the above variables were entered into a Cox proportional hazards regression model for: 1) pts discharged on a drug predicted to be effective, 2) all randomized pts (intention-to-treat), 3) pts with IHD. None of the VTns variables were significant independent predictors of arrhythmia recurrence or all-cause mortality. We conclude that significant clinical differences exist between groups of pts with different frequencies of VTns events. However, measures of VTns event frequency were not predictors of arrhythmia recurrence or mortality in this group of patients with known ventricular tachyarrhythmias.

3:15

719-6 Right Bundle Branch Block, ST Segment Elevation and Sudden Cardiac Death. Symptomatic, Asymptomatic and Intermittent Forms

Josep Brugada, Pedro Brugada [1]. *Hospital Clinic, University of Barcelona, Spain; Cardiovascular Center, Aalst, Belgium*

Objectives: We recently described a syndrome characterized by an electrocardiographic (ECG) pattern of right bundle branch block and persistent ST segment elevation in leads V1 to V3 in 8 patients (pts) suffering from aborted sudden death without structural heart disease. The objectives of the present study are to present new observations on this syndrome.

Methods: A total of 22 pts (mean age 40 ± 12 years) have been identified with the described ECG pattern. Of them, 17 were symptomatic with syncope and aborted sudden cardiac death. The other 5 pts were asymptomatic when first seen, however, 1 died suddenly after 6 years of follow-up without treatment and another received an implantable defibrillator after syncope. Subsequently, several episodes of ventricular fibrillation were terminated by the defibrillator in this pt. The other 3 pts remain asymptomatic without treatment. In 4 of 22 pts the ECG transiently normalized during follow-up.

Conclusions: The present observations demonstrate that there exist asymptomatic pts with this syndrome. These pts may become symptomatic during follow-up. It is difficult to set a therapeutic strategy for these asymptomatic pts at present. Our observations also show that the ECG can transiently normalize, suggesting that the disease is not based on a permanent structural cardiac abnormality, but rather on functional alterations of the electrical activity of the heart.

918 Clinical Studies of PTCA

Monday, March 20, 1995, 3:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

918-16 Hemostatic Puncture Closure Device Versus Regular Compression: A Randomized Study

Bernard Chevalier, Bernard Lancelin, Xavier Berthaux. *C.C. Marie Lannelongue, Le Plessis-Robinson, France*

In order to assess the efficacy of a new hemostatic puncture closure device (HPCD) (Angioseal*), 100 patients who underwent coronary angiography (75) or coronary angioplasty (25), using femoral approach, were randomized between HPCD placement (N = 52) or regular compression (C) (manual or mechanical pressure then pressure dressing) (N = 48). Clinical data, anticoagulation regimen, sheath size, delay of sheath removal, aPTT at the removal were not statistically different in both groups. Pts were randomized immediately before the sheath removal. A serial clinical follow-up was done until 24 hours and a systematic vascular ultrasonography (with pulsed and color doppler examination) was performed before hospital discharge. In the HPCD group, device placement was successful in all but one. Hemostasis was immediate in 67%; in 8 pts (15%) a light manual pressure was necessary (15 ± 10 min.) to stop the bleeding. Results in the two groups were:

	C	HPCD	p
Compression time (min.)	29.3 ± 23.2	2.3 ± 6.7	<0.001
Hemostasis delay (min.)	29.3 ± 23.2	4.4 ± 9.7	<0.001
Pressure dressing time (hr.)	20.7 ± 5.2	2.0 ± 7.9	<0.001
Ambulation delay (hr.)	20.2 ± 5.4	10.8 ± 7	<0.001
Minor local event (%)	23	7	<0.05

(=hematoma and/or rebleeding or small AV fistula)

We noted no major local event (false aneurysm, transfusion, surgical repair) in both groups. In HPCD group, ultrasonography showed the resorbable anchor in 96% which was correctly flushed against arterial wall in 94%.

Thus, HPCD use allows a dramatic decrease in compression and pressure dressing time, an earlier ambulation and a significant reduction in minor local complication.

918-17 Limitations of Percutaneous Interventions in the Treatment of Bifurcation Lesions Involving the Left Anterior Descending Coronary Artery

Brian A. Armstrong, Jean-François Tanguay, Kevin R. Kruse, James P. Zidar, Michael H. Sketch, Jr., Robert H. Peter, Harry R. Phillips, James E. Tcheng, Richard S. Stack. *Duke University, Durham, NC*

Serious complications may occur when intervention is unsuccessful in bifurcation lesions involving the left anterior descending (LAD) and first major diagonal (D), because of the large amount of involved myocardium. To determine this complication rate, we reviewed 82 consecutive cases, over a 3 year period, in which these lesions were attempted. Sixty-six percent of the subjects were male, and 37% had unstable angina. The mean age was 59 and the mean ejection fraction was 56%. Digital calipers were used to measure vessel minimum lumen (MLD) and reference diameters. For the LAD the final MLD was 1.81 mm and for the D 1.32 mm. The final percent mean diameter stenoses for the LAD and D were 41% and 45%, respectively. There were no significant differences in the rates of success or complication between groups treated with angioplasty only (N = 68) or directional atherectomy (N = 14). The in-hospital event-free success rate was 55%. The in-hospital complication rates were:

Recurrent Ischemia	16%	Ventricular Tachycardia	2%
Myocardial Infarction	14%	Stroke	2%
Bypass Surgery	12%	Death	1%
Repeat Procedure	4%	Composite	34%

Conclusion: LAD bifurcation lesion intervention is associated with a high in-hospital complication rate. Since these lesions are not amenable to stent placement or atherectomy with simultaneous protection of both vessels, these cases should be carefully evaluated before intervention, and bypass surgery should be considered as a treatment option.